A RESUMÉ OF THE PRINCIPAL DIAGNOSTIC FEATURES OF SUBDURAL HEMATOMA*

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CEREBRAL subdural hematoma is a lesion essentially of traumatic origin, occurring with increasing frequency in the mechanized era in which we live, producing symptoms at variable periods following injury and should be entertained as a possible complication of every craniocerebral trauma however trivial the accident may have seemed. As holds true for the advancements of our knowledge regarding any particular clinical entity, a few outstanding contributions concerning subdural hematoma have been responsible for the progress that has been made in the treatment of patients with this pathological process. It was in 1857 that Virchow[1] reviewed the previously proposed concepts relative to encapsulated hemorrhages in the subdural space and set forth his own thoughts regarding the pathogenesis of this lesion. This article appears to have influenced medical thinking for the next fifty years. In any event during this period it was quite generally held that blood clots disclosed in the cerebral subdural space were chronic in character and existed for the most part in idiots, the senile and the insane. In 1905 Bowen[2] presented a study of 72 cases of subdural hematoma reported in the literature between 1870 and 1900 and adduced evidence indicating that there was a causal relationship between trauma and this lesion. This excellent presentation of the subject remained relatively obscure and it was not until twenty years later that Putnam and Cushing[3] in 1925 focused the attention of the present generation of American surgeons on a pathological state that could be cured by appropriate surgical measures. Beginning in 1934 Munro[4] contributed a series of articles dealing with the pathogenesis, the diagnosis and the surgical treatment of the varied subdural collections that complicate craniocerebral injuries. His work has been important in that many problems relative to pathogenesis have been studied in considerable detail. Contributions by

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Frazier,[5] Peet and Kahn,[6] Jelsma,[7] Ingraham and Heyl,[8] Kaplan,[9] Kunkel and Dandy,[10] Leary[11] and others have advanced our knowledge concerning this lesion.

In striking contrast to the ideas held during the nineteenth century it is now well substantiated that trauma to the head is by far the most frequent etiological factor in the production of subdural hematoma, however, these lesions may be the result of spontaneous hemorrhage. I have observed examples of bleeding into the subdural space from cortical vessels implicated by a metastatic tumor of the brain, from ruptured cerebral aneurysms, from an arteriovenous malformation on the surface of the brain and from the region of subcortical abscesses. In most instances a moderately severe traumatic cerebral insult has been sustained with resultant contusion-laceration of a temporal and/or under-surface of a frontal lobe. Bleeding from lacerated vessels in one or the other of these damaged areas collects in the subdural space. Or a vein crossing from the cerebral cortex to the dura or leading into one of the dural sinuses may be torn with the production of a large blood clot. In some it is impossible even at autopsy to identify the source of the hemorrhage.

Recently established collections in the subdural space may consist of solid blood clots, blood admixed with cerebrospinal fluid or xanthochromic fluid in considerable quantity, the so-called subdural hydroma. Slowly a membrane is formed about the collection, that portion of the capsule on the dural side becoming relatively thick and quite vascular, whereas the membrane adjacent to the arachnoid is thin and devoid of prominent blood capillaries. The accumulated evidence indicates that coagulated blood usually undergoes liquefaction and, as pointed out by Gardner,[12] adjacent fluid (cerebrospinal) is drawn into the subdural collection producing an increase in the size of the intracranial mass. The clinical course of many patients suggests that the subdural mass may enlarge several days, weeks or sometimes months after the accident or, at any rate, symptoms put in their appearance at these late dates. Occasionally the clot is invaded by fibroblastic elements with a resultant organized mass. Yet patients in whom this has occurred may manifest the same delayed symptomatology as observed in those with encapsulated fluid collections. It seems unlikely that an organized blood clot could expand within a few days after being relatively quiescent for many weeks. Alterations in cerebral circulation in the vicinity of either an organized blood clot or the chronically encapsulated fluid collections

are more likely the explanation for the symptoms. In support of this concept specimens obtained at autopsy have shown obvious edema of the brain adjacent to an encapsulated fluid hematoma. This local edema could be logically assigned as the cause of the clinical signs which had been demonstrable. The organized blood clots and the more commonly encountered encapsulated liquid subdural collections are the lesions that have attracted the attention of neurological surgeons and concerning which excellent discussions have been recorded. These collections are not infrequently disclosed at operation, under the mistaken diagnosis of brain tumor. They are found in patients who have survived a mild to moderately severe head injury which in many instances has long since been forgotten. They have been termed chronic subdural hematoma, a designation entirely consistent with the duration of the lesion, however, the term should not be used to imply that this syndrome has a separate and distinct pathogenesis from the so-called acute subdural hematoma. The chronic state is merely a latent variant of the acute. Moreover the patients with chronic collections present symptoms and signs that are readily interpreted as representing a space-taking mass in the head and if not localizable by clinical findings, their position can be accurately determined by Dandy's method of ventriculography. The mortality from operative removal of these chronic lesions should be almost nil, therefore, further discussion concerning this entity need not detain us here.

The acute form and the subacute variety, if terms signifying duration of the lesion are permissible, are the commonest types of subdural hematoma. Some patients with this lesion die within a few hours following injury, some survive for several days in a state of continuous stupor, others rouse from stupor after three or four days only to lapse into coma in the next eight or ten days, and still others less severely injured become oriented two or three days after injury, remain so for two or three weeks albeit complaining of headache, then backslide into a confused state. While it is recognized that the subdural collections associated with these syndromes are often a single complication of a general brain insult and surgical removal may not favorably influence the eventual outcome in some that are severely injured, nevertheless, prompt recognition of the probability of such a lesion, the employment of appropriate diagnostic measures and the execution of judicious surgery will save many who otherwise would succumb.

During the past eight years there were admitted to the Kings County and Brooklyn hospitals 18,272 patients with varied types of craniocerebral injuries. Among these there were 289 instances of subdural hematoma. A study of 143 of these (five year period) has been previously reported.[13] Experiences with the entire group of 289 cases has led to the conclusion that an accurate diagnosis is seldom possible from the clinical features alone. In by far the majority of instances there were associated intrinsic brain lesions productive of abnormal physical signs which served to confuse the diagnostic issues. There are, however, a few clinical observations that are significant and are indispensable aids in arriving at a working diagnosis. These are: (1) the external evidence of trauma to the head and an estimation of the area receiving the maximum blow, (2) the state of consciousness, (3) the condition of the pupils, especially inequality, (4) weakness of an extremity or extremities of one side and (5) the status of the superficial and deep reflexes. Aside from the verification that a blow to the head has been sustained, the observations relative to the conscious state are most important. A short period of drowsiness or stupor, the consequence of the initial insult, followed by enduring orientation is seldom associated with subdural hematoma. However, a period of a day or two of confusion or drowsiness followed by several days to weeks of relative lucidity, during which the patient's intellect is mildly blunted, and then the supervention of progressive drowsiness to stupor, is highly suggestive of a subdural collection. Profound stupor following a craniocerebral injury, with or without physical signs indicative of a focal lesion of the brain, that continues longer than 48 to 60 hours also indicates that all is not well and the patient is in need of further diagnostic and therapeutic measures.

The condition of the pupils, especially inequality, has not been found to be as helpful a guide as reported by some. The pupils were recorded as unequal in 46 per cent, or 132 cases, of the series. In 29 per cent of these 132 cases, the larger pupil was on the opposite side to the subdural collection. In only 11 per cent was there found an unequivocal widely dilated pupil on the same side as the lesion. It is therefore evident that one may be misguided by attaching undue importance to the presence of unequal pupils.

Weakness of an extremity or extremities of one side was observed in 166 of the series. The hemiparesis was present on the same side as

the subdural hematoma in 64 of these. In other words, 22 per cent of the entire series of 289 cases had weakness of the extremities on the same side as the subdural hematoma. The presence of motor weakness of the extremities of one side following a craniocerebral injury does not necessarily indicate that the cerebral hemisphere of the side opposite the weakness is being compressed or otherwise functionally altered by a blood clot. If a hemiparesis be demonstrable, however, the possibility of an intracranial hematoma should be entertained and the diagnosis of such a lesion be established or excluded by small multiple cranial openings and/or ventriculography.

The reflexes, both superficial and tendon, are frequently found to be abolished shortly after a moderate or severe craniocerebral trauma. Babinski's sign may be present bilaterally. After recovery from the immediate effects of the insult the tendon reflexes may be obtained, although such bizarre reflex findings are frequently present that interpretation is difficult. In truth, observations relative to semi-purposeful or random movements of an extremity or extremities are more helpful in estimating disturbances in neural mechanisms than are refined tests.

Records of the pulse rate, respiratory rate, blood pressure and the cerebrospinal fluid pressure and its characteristics should be kept, but these singly or in combination are not to be considered as reliable indices for therapy. They are only a part of the entire clinical picture and are to be evaluated accordingly.

From this abbreviated account it would seem that time honored clinical findings derived from physical examination are wholly unreliable. In many instances this may be true and the evidence obtained from ventriculographic examination supersedes all the other observations. Although this form of examination is many times an indispensable aid in establishing or excluding the presence of a subdural hematoma or other intracranial blood clots, one does not inject air into the cerebral ventricles of every patient who is in an unconscious state. It is still important to arrive at a reasonable clinical diagnosis before subjecting the seriously injured to an operative procedure however minimal. Familiarity with the clinicopathological syndromes that are commonly encountered is therefore essential for differential diagnosis.

As previously alluded to, multiplicity of cerebral lesions in the same patient is one of the greatest obstacles in identifying the presence of a sizable blood clot that should be surgically removed. It is often impos-

sible to assign with certainty the etiological factors to obviously abnormal neurological states. The clinical features of the common syndromes may be elaborated upon to advantage. There should be no question regarding the therapeutic course to pursue in any patient who has sustained a cerebral insult with or without immediate loss of consciousness followed by a period of relative lucidity and this superseded by drowsiness, stupor and deepening coma. 'Tis true that exceptions are encountered; however, under these conditions the advent of drowsiness with or without focal signs is the signal for prompt surgical intervention. This is the story of slow arterial bleeding in the intracranial cavity. The blood clot may be located epidurally, subdurally or within the brain itself. The abnormal physical findings are often insufficient to localize the lesion with precision, therefore ventriculography is essential before any operative procedure is carried out. It is unwise and unsafe to attempt the removal of hematomas, be they epidural, subdural or intracerebral, through a traditional subtemporal decompression opening. If the subdural collection is not entirely liquid and cannot be evacuated through two small cranial openings then it is preferable to utilize a small bone flap made over the site of the lesion as demonstrated by the air studies.

There is a second clinical syndrome that calls for prompt and precise diagnosis followed by appropriate surgery according to indications. The following is an illustrative story: A patient who has sustained a moderately severe craniocerebral injury is admitted to the hospital shortly after accident. He is drowsy, disoriented and non-coöperative. The pupils are equal, the extremities are moved about equally well and no abnormalities in reflexes are demonstrable. Slowly over a period of eight to twelve hours stupor supervenes and one pupil is found to be slightly larger than its fellow. Pinching the pectoral muscle border or application of other painful stimuli evokes acceleration and increased depth of respirations and transitory skeletal muscle hypertonicity with all the extremities in a fully extended and rigid posture. Shortly, this rigid state, or so-called decerebrate rigidity, becomes almost continuous, the breathing is labored and forceful, the jaw set and the facial muscles contracted, simulating somewhat the sardonic grin of tetanus. The temperature rises and the pulse becomes accelerated. The skin is flushed, at first dry and hot, later the patient is bathed in his own sweat. Such a course of clinical events has been observed in association with a wide

variety of lesions: epidural, subdural, intracerebral or intraventricular hemorrhage, diffuse small hemorrhages throughout both cerebral hemispheres often associated with contusion/laceration of one or both frontotemporal regions, and/or hemorrhages in the upper part of the hind brain. The pertinent point relative to the present discussion is that a large subdural hematoma may be the cause of this clinical syndrome. While it is known that the decerebrate or decorticate attitude, regardless of the causative factor, is a bad prognostic sign, nevertheless, we have had a number of recoveries following the removal of large blood clots in patients manifesting these clinical features. It has been observed that inequality of pupils and the decerebrate attitude are more commonly associated with large supratentorial hematomas than are equally constricted pupils and the decerebrate attitude which are not infrequently found in association with hemorrhage within the upper hind brain. In all events the appearance of decerebrate rigidity in any patient who has sustained a head injury calls for ventriculographic examination unless a surface hematoma be disclosed in the making of bilateral openings for purposes of this examination. Further surgery is carried out in accordance with the information derived by inspection through the cranial burr openings and/or the aerographic study.

The third group of patients with craniocerebral trauma complicated by subdural collections comprise those that remain confused, drowsy or stuporous from the time of injury until the hematoma is surgically removed and/or death ensues. The obvious lesions of the brain disclosed in those that come to autopsy are single or multiple surface contusions or lacerations with regional edema. As previously stated a surface vessel, often a vein, has been torn at the site of the cerebral contusion and bleeding has occurred into the subdural space. Approximately two-thirds of all subdural hematomas are the result of this type of lesion. The clinical course of patients so injured is quite variable, dependent no doubt on the pathophysiological alterations within the brain itself. At least the marked changes in temperature, pulse rate, respiration, etc. suggest primary dysfunctions of the vegetative system. On the somatic side, hemiparesis or hemiplegia are not uncommon. Here again the exact role played by the subdural collection in the production of motor weakness of the extremities is difficult of evaluation. Often a lesion within the brain appears to be the more important. Be this as it may, patients severely injured frequently die within twelve to thirty-six hours follow-

CHART I

Number of subdural hematomas operated 227
Number of subdural hematomas non-operated but verified at autopsy.. 62
Total number of subdural hematomas (operated and non-operated).... 289

Time elapsed between Injury and Operation	*Total*	*Recovered*	*Died*	*Mortality*
2 to 24 hours	51	9	42	82%
1 to 7 days	65	34	31	48%
7 to 14 days	48	36	12	25%
14 to 21 days	33	26	7	21%
21 to 28 days	11	8	3*	27%
Over 28 days	19	17	2	11%
Totals	227	130	97	42%

* One of these had a subdural collection associated with multiple metastatic foci of infection in the brain.

ing the accident. Surgical removal of a subdural hematoma carried out during this critical period seldom alters the situation in a favorable manner. In fact we have come to believe that the removal of hematomas during the first twenty-four hours after injury is seldom justifiable. Chart I clearly illustrates the reasons for this position.

This chart shows the progressive decrease in the mortality rate following operation in those that were successfully carried through the critical period after injury. The sixty-two patients who died without operation represent those admitted to the hospital in a moribund state. Many of the patients from whom subdural hematomas were removed during the 48 hour period following injury presented evidence of severe brain damage. Among these, operation was a "last-resort" affair in many instances.

If the employment of supportive measures will not tide the patient over this critical period, then little should be expected from surgical intervention. Those less seriously injured usually weather the storm. However, if after a few days of progressive improvement there ensues drowsiness or untoward alterations in vital signs, the presence of a subdural hematoma should be established or excluded by making multiple small cranial openings and/or ventriculography. If a hematoma is disclosed by these examinations it should be promptly removed regardless of its anatomical position.

SUMMARY

It has been well established that in the majority of instances trauma is the cause of cerebral subdural hematoma. The varied alterations that take place in the size and consistency of many of these blood clots is still not well understood. Furthermore, the role played by cerebral edema adjacent to a chronic encapsulated hematoma in the production of symptoms and signs is also in need of further consideration. The clinical manifestations attending craniocerebral injuries complicated by subdural hematomas are difficult of interpretation. Multiplicity of lesions within the intracranial cavity is the rule rather than the exception. A few clinical syndromes are encountered, however, that suggest the possibility of a subdural collection. Whenever suspicion arises regarding the presence of such a lesion the diagnostic issues should be clarified by multiple small cranial openings and/or ventriculography. Except under circumstances as described the surgical removal of a subdural blood clot during the first 24 to 48 hours following injury is of questionable value. Patients seriously injured should be given supportive treatment during the critical phase of their illness and surgical therapy instituted only after the vital signs indicate a degree of stabilization.

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